

July 23, 1993

Richard J. Londergan, Ph.D.  
Senior Program Manager  
ENSR Consulting and Engineering  
95 Glastonbury Boulevard  
Glastonbury, CT 06033

Dear Dr. Londergan:

Thank you for your peer reviewer comments for the following documents:

- "Lead and Cadmium Exposure Study, Galena, Kansas" (draft final report);
- "Lead Exposure Study, Madison County, Granite City, Illinois" (draft final report);
- "Management of Children with Slightly Elevated Blood Lead Levels," by Renate D. Kimbrough, M.D., et al (manuscript).

They will be forwarded to the Division conducting the study for its evaluation. We appreciate your taking the time from your busy schedule to assist us and look forward to working with you again in the future.

If you do not receive a check for your professional services within 60 days, or if I may be of any other assistance, please give me a call (404-639-0708).

Sincerely yours,

Donna M. Rossie  
Peer Review Coordinator  
Office of the Associate  
Administrator for Science

Agency for Toxic Substances and Disease Registry  
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**CONFLICT-OF-INTEREST FORM**

To the best of my knowledge and belief, there are no relevant facts or circumstances that could give rise to any apparent or real personal or organizational conflict of interest regarding my work under this assignment. If any actual or apparent conflict of interest is discovered during the performance of this task, I will immediately notify the Associate Administrator for Science and will make a full disclosure in writing. In addition, I certify that neither I nor my spouse nor minor children have any financial or other interests that will cause an actual or apparent conflict of interest with my review of the document(s) specified below.

If I am a state or local government employee, I certify that: (1) my position is not being funded by any Federal grant; (2) my accepting remuneration does not conflict with state or local laws; and (3) this work will be performed outside my regular tour of duty.

I understand that peer reviewers will be identified by name and affiliation. Individual peer reviewer comments may be released by name under the Freedom of Information Act. Individual or summary-peer reviewer comments may be included in study reports.

Name of document under review: "Lead Exposure Study, Madison County, Granite City, Illinois"

Protocol or Final Report: Draft Final Report

Institutions involved: Illinois Department of Health

NL Industries/Taracorp Site

Richard J. Londergan, Ph.D.

Printed name

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**"LEAD EXPOSURE STUDY, MADISON COUNTY, GRANITE CITY, ILLINOIS"**

**PEER REVIEWER COMMENT FORM**

**July 1993**

**Richard J. Londergan, Ph.D.**

**Name** \_\_\_\_\_

- 1. Were the study objectives clearly stated and appropriate? Why or why not?**

*Study objectives and context were clearly stated. As part of the multi-state exposure study, overall objectives reflect careful planning and good definition.*

- 2. Was the study design appropriate for the study objectives? Why or why not?**

*The study design left a great deal to be desired, in that a suitable "control" population was not identified. This deficiency was clearly recognized and thoroughly addressed in the report, but it still led to many unresolved questions concerning results.*

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Name \_\_\_\_\_

3. Was the data analysis conducted in such a way to appropriately address the objectives of the study? Why or why not?

Given the limitations noted above, the data analysis was quite appropriate. ~~the~~

Analyses were focussed on the stated objectives and issues of concern, and were complete without a lot of extraneous data manipulation.

4. Were the selected methods appropriate for the chosen study objectives? Why or why not?

The selected statistical methods were appropriate for the data sets, were applied and documented appropriately, and were interpreted in a knowledgeable and logical fashion. Problems or unexpected results were discussed candidly, ~~straightforwardly~~ and approaches for addressing study objectives despite those problems were devised (though not always successful).

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Name \_\_\_\_\_

5. Were the conclusions and recommendations appropriate and complete?

Discussion of results and conclusions drawn were complete, and generally defensible, with the exception of a few unsupported statements, except for one central issue of interpretation:

The statistical result that, based on regression analysis, only 3 percent of the variance in blood lead values is "explained" by variations in soil lead, does not mean that soil lead only contributed 3

6. Any overall comments on the final report?

percent of  
total exposure.

The final report, overall, is excellent. It provides a careful, balanced, well-written presentation of the approach and results of the study.

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Name \_\_\_\_\_

7. Any comments on ATSDR's peer review process? *no*

8. Any other comments?

Would you like a copy of the final report? Yes ( ☒ ) No ( ☐ )

Select the appropriate category below (List recommended changes or reasons for not recommending):

A. Recommend ( ☐ )

B. Recommend with Required Changes ( ☒ )

*see item 5*

C. Not Recommended ( ☐ )

Signature

*Richard J. Londergan*

Date

*7/20/93*

multiplying this ratio by the dust lead concentration. The "dust load" transformation used in this report combines the lead concentration and the amount of dust present in the house in one variable.

The concentration of lead in drinking water was determined in a first draw sample by graphite furnace atomic absorption. Cadmium similarly was determined in house dust and soil by ICAP emission spectroscopy and in water by graphite furnace atomic absorption. The limit of detection for lead in house dust was 20 ppm (mg/kg), for soil  $\leq 20$  mg/kg and for drinking water  $\leq 2$  ppb ( $\mu\text{g/L}$ ). The limit of detection for cadmium in house dust was 2 ppm (mg/kg), soil 1.0 ppm (mg/kg), and for drinking water  $\leq 0.5$  ppb ( $\mu\text{g/L}$ ).

see  
p 34

#### Reporting of results to participants

The participants were informed of their individual clinical and environmental results by letter. The results of the clinical tests were presented at a public meeting in the Spring of 1992 without revealing the identity of the participants to reassure residents and encourage parents of untested children to have them tested. All families with at least one child with a blood lead level of  $0.48 \mu\text{mol/L}$  ( $10 \mu\text{g/dl}$ ) or above were visited, and potential sources of lead in the immediate environment of the child were identified for the guardians. The guardians were also instructed in nutrition, in personal hygiene of the children, and in reducing exposure through housekeeping and minor remediation of trouble spots in or outside of the homes.

Blood lead levels were also determined in 214 youths (ages 6-15 years), 111 males and 103 females, and in 47 males and 76 females over the age of 15. Thus, 827 blood lead determinations were made in all. The arithmetic mean blood lead levels for the youngest age group (between 6 and 71 months of age) was 0.33  $\mu\text{mol/L}$  (6.9  $\mu\text{g/dl}$ ) with a range of 0.03-1.94  $\mu\text{mol/L}$  (0.7-40.2  $\mu\text{g/dl}$ ). In this group, 78 children (16%) had elevated blood lead levels of 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) or above. For the children between the ages of 6 and 15 years, the arithmetic mean blood lead level was 0.21  $\mu\text{mol/L}$  (4.4  $\mu\text{g/dl}$ ), the range <0.03-0.90  $\mu\text{mol/L}$  (<0.6-18.8  $\mu\text{g/dl}$ ). In this group, 8 individuals had blood lead levels of 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) or above.

Among a total of 101 non-white children under the age of 6 87% were of African-American descent. Of these children, 16% had elevated blood lead levels. The arithmetic mean blood lead levels of all white children under 6 years of age was 0.32  $\mu\text{mol/L}$  (6.8  $\mu\text{g/dl}$ ) and for the children of African-American descent, the arithmetic mean was 0.35  $\mu\text{mol/L}$  (7.4  $\mu\text{g/dl}$ ). Thus, the blood lead levels of children of African-American descent were quite similar to those of the white children ( $t = -1.1$ , NS) and 19% had blood lead levels of 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) or above. These two groups of children were, therefore, combined in the analysis.

Among the children 6 years and older, 17 boys and 16 girls of African-American descent participated in the study. Their arithmetic mean blood lead levels were 0.20  $\mu\text{mol/L}$  (4.2  $\mu\text{g/dl}$ ) and 0.23  $\mu\text{mol/L}$  (4.7  $\mu\text{g/dl}$ ), respectively. None of these

children had blood lead levels of 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) or above.

The arithmetic mean blood lead levels of participants greater than 15 years of age was 0.17  $\mu\text{mol/L}$  (3.6  $\mu\text{g/dl}$ ) with a range of <0.03-0.86  $\mu\text{mol/L}$  (<0.6-17.9  $\mu\text{g/dl}$ ). The blood lead levels above 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) in 3 male participants had <sup>likely</sup> resulted from occupational exposure or hobbies. The total number of 43 white adult males had an arithmetic mean blood lead level of 0.28  $\mu\text{mol/L}$  (5.8  $\mu\text{g/dl}$ ) and included the 3 males with elevated blood lead levels. Elevated blood lead levels did not contribute to the arithmetic mean blood lead level of 69 adult white females. Their arithmetic mean blood lead level was 0.12  $\mu\text{mol/L}$  (2.4  $\mu\text{g/dl}$ ). Among the adult females, 14 were pregnant at the time the blood specimen was drawn. Their blood lead levels ranged from <0.03  $\mu\text{mol/L}$ -0.16  $\mu\text{mol/L}$  (<0.6-3.4  $\mu\text{g/dl}$ ) with an average of 0.03  $\mu\text{mol/L}$  (1.6  $\mu\text{g/dl}$ ). Three adult males and 7 adult females of African-American descent also participated in the study with arithmetic mean blood lead levels of 0.18  $\mu\text{mol/L}$  (3.8  $\mu\text{g/dl}$ ) and 0.17  $\mu\text{mol/L}$  (3.5  $\mu\text{g/dl}$ ).

In the youngest age group, 78/490 (16%) had blood lead levels above 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ); however, 46 of these (9% of the 490) had blood lead levels between 0.48-0.72  $\mu\text{mol/L}$  (10-15  $\mu\text{g/dl}$ ) and only 5 (1% of the 490) were above the pre-1991 level of concern of 1.21  $\mu\text{mol/L}$  (25  $\mu\text{g/dl}$ ) of the CDC (Table 5). A total of 61 children with blood lead levels above 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) and some of their siblings donated a second blood specimen.

### Environmental data

A total of 34% of all participants did not know the age of the house in which they were living. Among the 412 children under 6 with blood lead levels of less than  $0.48 \mu\text{mol/L}$  ( $10 \mu\text{g/dl}$ ) data on the age of the houses was available for 278. Of those children, 196 or 70% lived in houses that were built before 1950. Of the 78 children with blood lead levels of  $0.48 \mu\text{mol/L}$  ( $10 \mu\text{g/dl}$ ) or above data on the age of the houses was available for 43. Of those children, 35 or 81% lived in houses built before 1950. Of the children with elevated blood lead levels who lived in houses built after 1950, one child lived in a mobile home and the father was involved in lead related activities. The other houses were built between 1950 and 1970 and remodeling activity or refinishing of furniture had taken place between 1990 and 1991.

Lead levels measured in paint and in soil of the houses are given in Tables 9a and 9b. Houses in which children with elevated blood lead levels lived were not clustered. However, these children were more likely to live closer to the smelter (Figure 1). Of the children under 6 with blood lead levels below  $10 \mu\text{g/dl}$  ( $0.48 \mu\text{mol/L}$ ), 16% percent lived in sampling area 1, 43% in sampling area 2, 24% in sampling area 3 and 16% in sampling area 4. Among the children whose blood lead levels were above  $10 \mu\text{g/dl}$ , 27% lived in sampling area 1, 53% lived in sampling area 2, 12% lived in sampling area 3 and 8% lived in sampling area 4. Many of the children lived in houses with high paint lead

only 20 of 388<sup>32</sup> households

concentrations in one or more of the areas measured (Table 9a). Either recent renovation or poorly maintained houses seemed to contribute to the exposure of the children. When the houses were in good condition, increased lead exposure was not as much of a problem.

52? Overall, about 50% of the families, had done some repair work or renovations on their houses in 1990 or 1991. For families with children under 6 whose blood lead levels were below 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ), 48% had done some work on their house in the last year and [REDACTED] did not. In contrast, 63% of the families whose children had blood lead levels above 0.48  $\mu\text{mol/L}$  (10  $\mu\text{g/dl}$ ) did some refurbishing in the last year while 38% did not. The difference was statistically significant ( $p < 0.02$ ).

In many yards, the lead concentrations in soil were above background levels which locally ranges from below the limit of detection of 1 ppm (mg/kg) to 200 ppm (mg/kg). The mean soil lead level for the 376 analyzed soil samples was 450 ppm (mg/kg) with a range of 37 ppm (mg/kg) to 3010 ppm (mg/kg) (Table 9b). A total of 39 split samples were also analyzed. The concentration of lead in these soil samples ranged from 106 to 1610 ppm (mg/kg). The average difference between the primary and the duplicate sample was 89 ppm (mg/kg).

It is evident from Tables 9a and 9b that there are some very high environmental lead values. For example, the minimum dust lead values is 5.2 mg/kg (ppm), the maximum value is 71,000 mg/kg (ppm), and the standard deviation is nearly four times as great

as the mean. Most of the other data were also not normally distributed. Log-transformed data was, therefore, used for most of the statistical analyses.

2 A total of 376 composite soil samples were also analyzed for cadmium. The arithmetic mean cadmium concentration in soil was 3.1 ppm (mg/kg) with a standard deviation of 1.37. Cadmium was not detected in 8 soil samples at a limit of detection of 3 ppm (3 mg/kg) and all but 7 soil samples were below 6 ppm (mg/kg). The concentrations of cadmium in soil generally ranges from 0.3-11 ppm (mg/kg) (Page and Bingham, 1973; Lund et al., 1981). Thus, cadmium concentrations are within the background range of concentrations found by others. (see p. 16)

Lead in drinking water was below the limit of detection of the analytical method of 2  $\mu\text{g/L}$  (ppb) in 62% of the samples of 374 households. A total of 86% of the samples had levels of 5  $\mu\text{g/L}$  (ppb) or less and 97% were below 15  $\mu\text{g/L}$  (ppb), the present USEPA action level. In 13 instances, levels of lead in drinking water were higher with a range of 15.4-95.5  $\mu\text{g/L}$  (ppb). The study participants using this water did not have elevated blood lead levels. The correlation between the log water measure and log blood lead was very low ( $r = 0.07$ , N.S.).

The concentrations of cadmium in 374 drinking water samples were below the limit of detection of 0.1  $\mu\text{g/L}$  (ppb) in 322 samples and the maximum concentration detected was 9.9  $\mu\text{g/L}$  (ppb). Only 11 samples were above 2  $\mu\text{g/L}$  (ppb). In a survey of 969 community water supply systems in the United States the (see p. 16)

2  
good condition had a mean soil lead concentration of 287 ppm (mg/kg). The mean soil lead concentration for houses in fair condition was 361 ppm (mg/kg) and for houses in poor condition it was 459 ppm (mg/kg). Building condition differs from other potential confounders of the composite soil lead/blood lead association in that the condition of the house is not likely to be a pathway for soil lead exposure. It is one of the few confounders of the soil lead, blood lead relationship that can be controlled for statistically.

Cigarettes per day

(I disagree)

In this data set smoking is associated with blood lead. The number of smokers ( $r = 0.16$ ;  $p < 0.01$ ), and the number of cigarettes smoked per day ( $r = 0.23$ ;  $p < 0.01$ ) both predict blood lead to a degree. However, the number of cigarettes smoked per day is also correlated with "dust load" ( $r = 0.15$ ;  $p < 0.01$ ); but not with dust level (i.e. the weight of the dust sample divided by the area vacuumed,  $r = 0.005$ ;  $p = 0.92$ ). The number of cigarettes smoked per day is also correlated with composite soil lead ( $r = 0.17$ ;  $p < 0.01$ ), distance from the smelter, parents' education ( $r = -0.34$   $p < 0.01$ ), income ( $r = -0.20$ ;  $p < 0.01$ ) and outdoor paint lead ( $r = 0.11$ ;  $p < 0.02$ ). Furthermore, smokers in houses without air conditioning smoked 35.4 cigarettes per day, while 17.5 cigarettes per smoker were smoked in houses with air conditioning ( $t = -3.8$ ;  $p < 0.01$ ). More cigarettes were smoked in houses in poorer condition ( $F = 17.2$ ,  $df = 2$ ,  $p < 0.01$ ); and in older houses ( $r = 0.16$ ;  $p < 0.01$ ). It is impossible to determine

A number of variables predicted blood-lead levels in young children. These included condition of the house, lead in paint, lead in dust, lead in soil, smoking of the parents, proximity to the defunct smelter, education and income of the parents, and behavioral factors of the children such as hand-to-mouth activities. Comparing these factors showed that they were all correlated with each other. Only about 40% of the exposure could be accounted for in our data analyses. Of these 40%, lead from soil appears to make a very minor contribution, as an upper-bound at most 3% while the condition of the house and the amount of lead in paint may be responsible for as much as 11%.

Most of the important variables in this study such as education and income of the parents, lead in paint, soil-lead, dust lead, behavior variables, smoking and air conditioning are all highly correlated. Thus, correlations, t-tests and Chi-square tests if taken out of context may be misleading. Furthermore, confounding can not be adequately controlled for in the present data set. Many important behavioral variables may affect the degree of exposure to house dust that serves as the primary pathway of exposure for soil lead and house paint lead in small children. Very small but statistically significant differences of a few percent of the variance contributing to blood lead levels have no clinical significance. We attempted to determine, by step-wise regression of 22 variables, what the overall contribution of these variables to lead exposure was. However, as some variables were added to the analyses other

inability to account for 60% of lead uptake underscores that point.

- Education of the parents about the lead hazards in their individual homes and suggestions for remedial action and behavior has a favorable impact on the children's blood lead levels.
- High levels of lead in soil had little effect on blood lead levels accounting for 3% of the variance in blood lead.
- Our findings suggest that removal of soil as a remedy will generally not have a beneficial effect on children's blood lead levels.
- Many of the houses inhabited by our study population had high lead paint levels. The lead from the paint particularly in houses that were poorly maintained or had recently undergone repair contributed to increased exposure.
- High concentrations of lead in paint in well-maintained houses did not contribute noticeably to lead exposure. Many of the children with low blood lead levels lived in houses in good condition even with very high lead paint levels.

Not supported. Effect unknown  
(not "no effect")

## RECOMMENDATIONS

Reducing blood lead levels in young children is best accomplished through education of the children and their caretakers and through reducing exposure to paint with high concentrations of lead. Since house dust is the primary transport mechanism through which children are exposed keeping houses clean and well-maintained is the most important factor in reducing lead exposure. Removal of lead contaminated soil will not reduce blood lead levels in children in the Granite City area. Soil removal alone over extended residential areas should generally not be recommended as a solution to reducing lead exposure if lead paint problems are not addressed. Soil removal as the sole remedy should be the exception rather than the rule.

not supported (effect unknown)